Brainstem Strokes

Introduction

Having discussed the lesions of cerebral circulation, we now turn our attention to brainstem strokes. Brainstem strokes are more challenging than those of the cerebral circulation because you have both nuclei and tracts to contend with, and there aren't obvious vascular territories as there are with strokes of the cortex. Every part of the brainstem has at least two (usually more) arteries supplying each side. There is no simple way to break this down, except to say that every branch of the posterior circulation has a syndrome associated with it. And so, this lesson works its way up the brainstem, just as this summary table works up the page. Start at the bottom, and work your way up the table row by row.

ARTERY	SYNDROME	DESCRIPTION
Paramedian branches of basilar	Median midbrain (Weber superior) (Benedikt inferior)	Ipsilateral oculomotor palsy (CN III) Contralateral DCMLS hemisensory loss body and face (Weber, superior colliculus) Contralateral hemiplegia (Benedikt, Inferior colliculus) Contralateral cerebellar ataxia
SCA	Superior cerebellar	Ipsilateral cerebellar gait ataxia, limb ataxia, and vertigo
Basilar	Median pons	Locked-in syndrome Quadriparesis, paralysis of the facial muscles, 5/6 extraocular muscles work, the eyelid can lift Loss of sensation of the entire body, facial sensation may remain intact
AICA	Lateral pons	Ipsilateral CN VII (paralysis of the face, decreased salivary output, loss of taste on anterior 2/3 of tongue Vertigo, vomiting, nystagmus, deafness Ipsilateral loss of pain and temp of the face Contralateral loss of pain and temp of the body Ipsilateral Horner's syndrome
PICA	Lateral medulla	Ipsilateral IX and X (impaired gag reflex, impaired swallowing reflex, loss of dysphagia, dysarthria) Vomiting, nystagmus Ipsilateral loss of pain and temp of the face Contralateral loss of pain and temp of the body Ipsilateral Horner's syndrome
ASA	Median medulla	Contralateral hemisensory loss Contralateral hemiplegia Tongue deviation towards the side of the lesion
Vertebral arteries	Spine	None

Table 7.1: Brainstem Lesion Locations and Their Associated Syndromes

Some Rules

For those *tracts going to or from the spinal cord*—DCMLS, STT, corticospinal tract—there are some rules.

There are no nuclei to be damaged in the brainstem—those are either in the cortex or spinal cord. So, damage can occur only to tracts. Good news, the three systems you care about, except for a tiny portion of the inferior medulla, all three tracts—corticospinal, DCMLS, and STT will have already crossed, giving you congruent motor and sensory deficits. Almost all (except for medial medullary syndrome) will show findings on physical exam that are contralateral to the lesion.

The STT synapses and decussates immediately at the level of the spinal cord. If the STT is lesioned in the brainstem, the entire tract is lost. And because it has already crossed, pain and temperature defects resulting from brainstem-level lesions are **always contralateral** to the lesion.

The DCMLS ascends on the ipsilateral side has the sensory neuron then synapses and decussates at the distal medulla. Therefore, only the most distal medullary lesions can cause ipsilateral deficits. For simplicity, in the brainstem, consider all DCMLS lesions to be like those in the STT: The entire tract has already crossed, so the entire tract is lost if it travels into a brainstem lesion. Proprioception, vibration, and touch defects are **always contralateral** to the lesion.

The corticospinal tract descends and decussates at the distal medulla. While the ascending DCMLS will have already completely crossed before hitting a brainstem lesion, none of the corticospinal tracts will have crossed, except in cases of the most distal medullary lesions. The entire tract will run into a lesion of the brainstem and be lost. Losing a tract means **upper motor neuron** symptoms, and the side affected will **always be contralateral** to the lesion.

We made you do that exercise instead of just saying, "*all tracts will always be contralateral,*" because visualizing the tracts passing through the lesion and being lost will revitalize your memory from Motor and Sensory Tracts #3: *Spinal Cord Lesions*.

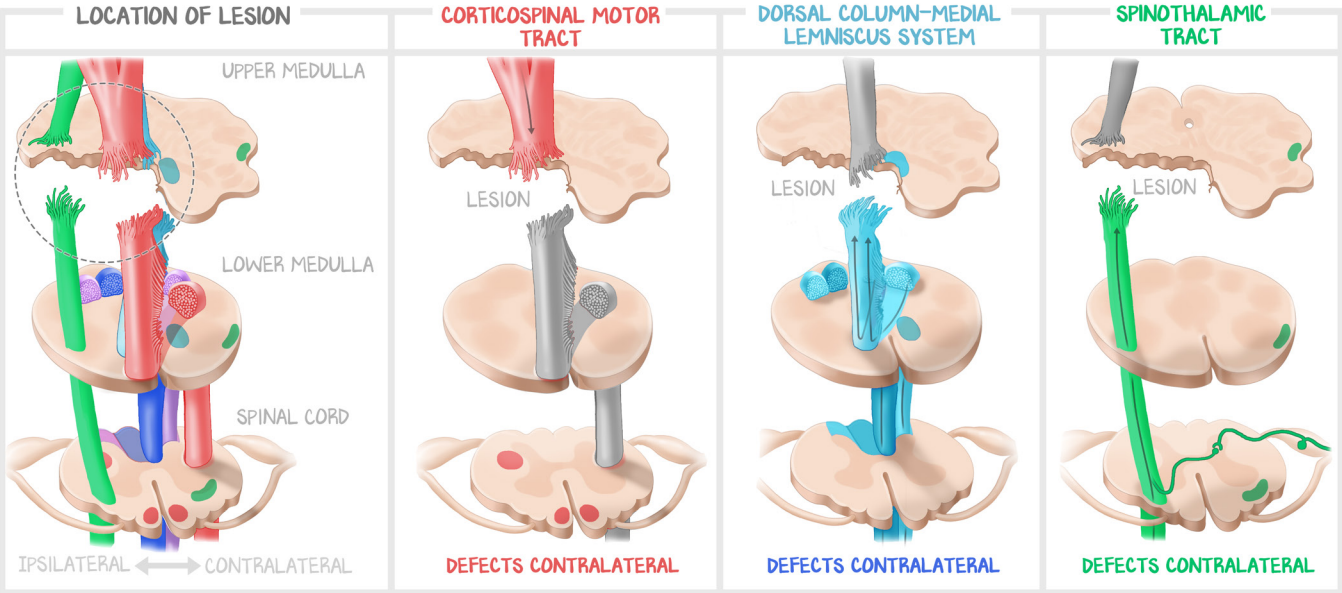

Figure 7.1: Spinal Tracts Lesioned in the Brainstem Are Contralateral
Axons of all tracts cross to the side contralateral to their origin. STT axons cross at the vertebral level at which they enter—synapse and cross immediately. The corticospinal tract and the DCMLS axons cross at the most distal—most inferior—medulla. We are isolating this lesson to the brainstem only (no cerebral lesions, no spinal cord lesions), so because all the spinal cord tracts will have already crossed, a loss of the tract means the loss of communication (whether ascending or descending) to the entirety of the contralateral body. It isn't as simple for the corticobulbar tracts (from the cortex to the cranial nerves that innervate the face) but is super reliable as it pertains to the body.

For the *peripheral nerves of the cranium*, there are some different rules.

The loss of a **brainstem nucleus** results in **ipsilateral** symptoms. Because we're specifically talking about brainstem strokes, there won't be any cortical lesions. The **corticobulbar tracts**, the motor tracts that take up the medial third of the crura cerebri of the cerebral peduncles, **cross at the level** of their lower motor neurons. This means that if you have lesions of the tract, the defect will be on the **contralateral side.** However, because the vascular lesion syndromes don't involve the tracts but rather the nuclei, the syndromes you have to know will have symptoms **ipsilateral** to the brainstem lesion. When there is a contralateral lesion of a cranial nerve tract, we will call it out specifically. Just as there is a corticobulbar (from the cortex to the brainstem) motor tract that decussates at the level it enters the brainstem, so too are there brainstem equivalents of the DCMLS and STT that decussate at the level they enter the brainstem.

That means that if **tracts are affected**, the symptoms will **always be contralateral** to the lesion, whether it's a lesion of the corticospinal tract or corticobulbar tract. And if there are **any ipsilateral symptoms**, there must be a **lesion of a brainstem nucleus**.

Anterior Spinal Artery Truth

We go with the flow with the other syndromes in this note set, but take issue with what others have been teaching about medial medullary syndrome. This section demonstrates why regurgitating memorized facts is not the path to mastering health and disease. Every review resource says that medial medullary syndrome is a lesion of only one side of the medulla and involves ipsilateral tongue deviation, contralateral hemiparesis, and contralateral hemisensory loss. Everyone is wrong. And it is obvious according to either physiological logic or data from the limited case reports available.

The **anterior spinal artery** arises from the two vertebral arteries. It courses between the medullary pyramids. The medullary pyramids represent the decussation of the DCMLS and corticospinal tract. Damage to the anterior spinal artery in the medulla is known as **medial medullary syndrome**, and has a dramatically different presentation than when it is damaged in the spine. Medial medullary syndrome is so rare because the anterior spinal artery forms from both the vertebral arteries and is continuous with the anterior spinal artery of the spinal cord, which receives multiple tributaries through the aorta. With all of that blood supply and all those anastomoses, there is essentially no distal or proximal—any lesion of the artery will affect only a sliver of the medulla, with perfusion being maintained in the rest of the medulla because of all the anastomoses. This means that the symptoms of medial medullary syndrome are likely to reflect the **loss of spinal cord tracts**, not medullary symptoms.

Medial medullary syndrome was once called "inferior alternating syndrome." "Inferior" because the affected segments only provoke symptoms in the lower extremities. "Alternating" because it causes **both contralateral and ipsilateral symptoms**. How can something provoke both contralateral and ipsilateral symptoms? There are two explanations. The first is that the medulla is where both the DCMLS and corticospinal tract cross, and those crossings are topographic. Some axons cross into the lesion and disintegrate, some cross out of the lesion and are spared, and others don't cross in time, so they collide with the lesion and disintegrate.

Medial medullary syndrome is commonly taught as the contralateral loss of general touch sensation, contralateral loss of motor function, and ipsilateral loss of the hypoglossal nerve. That's because there haven't been enough cases to really know what the disease looks like. ASA lesions account for < 1% of all brainstem strokes, so a "classic" syndrome is impossible to define. And when you look at the "half and half" maps (like the one below), you forget that there is only one anterior spinal artery, not left and right ones. Therefore, the second reason there are both ipsilateral and contralateral lesions is that there is **one anterior spinal artery** that affects both sides of the medulla at once, just like in anterior spinal artery syndrome in the spinal cord.

Because the vascular supply comes from the anterior and the structures are arranged anteroposteriorly—the corticospinal tracts being the most anterior, followed by the medial lemniscus of the DCMLS, then the hypoglossal nerve—one would expect to see the highest incidence of hypoglossal nerve symptoms, followed by sensory symptoms, then motor symptoms. But that's not what happens.

The **hypoglossal nerve** is only in jeopardy where the fourth ventricle closes and the medulla transitions to the spinal cord. That's approximately 5 mm of vulnerability (the medulla is about 3 cm in total length). Because it is the farthest away from the blood supply (it is posterior and in front of the ventricle), it IS the most vulnerable structure. And yet, hypoglossal dysfunction (tongue deviation) is the **least encountered symptom** because ASA lesions tend not to occur in those 5 mm that the hypoglossal nerve is perfused by the anterior spinal artery.

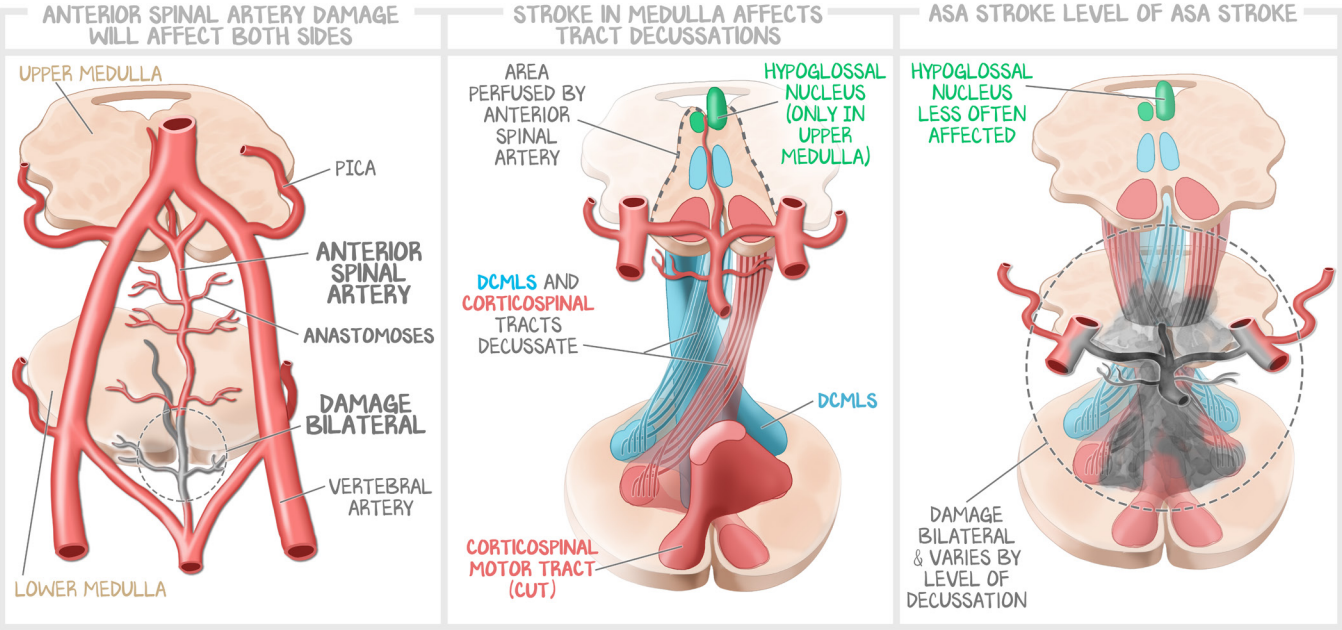

Figure 7.2: Why the Classic Lesion Isn't Classic
The one anterior spinal artery feeds both sides, so damage is most likely to be bilateral. Even if the damage is unilateral (as caused by a terminal, unnamed branch of the anterior spinal artery), the hypoglossal nucleus is unlikely to be affected, despite being the most vulnerable as it's located at a minuscule stretch of the medulla (at which anatomic variants more likely salvage the nucleus than compromise it). There may also be a lesion of the decussation, at which both DCMLS and corticospinal tract axons cross (making an alteration of symptoms including motor and sensory more likely). With so many possible variations, there is no single "medial medullary syndrome," but rather a wide variety of medial medullary syndromeS, with significant variation in the presentation.

Whenever only the DCMLS and corticospinal tracts are at risk, the anteroposterior concept works out more often than it doesn't (i.e., there are more sensory symptoms than motor symptoms), but there have been conflicting case studies that imply that simple lack of perfusion cannot be to blame. In one case series, the most common symptoms were hemisensory deficits. In another, the most common symptom was hemiplegia. Some were motor only, some were sensory only, and a handful were both. Regardless of whether they were motor- or sensory-predominant, **most were contralateral,** and only a **few were bilateral**. Almost none saw hypoglossal dysfunction.

After doing the research, we were met with a conundrum—regurgitate the same old stuff as always (because you are so unlikely to see it in life, and so you're very likely to see it on a licensing exam), or spend a long time showing you that it **can present as anything**—sensory, motor, tongue movement, ipsilateral or contralateral—**because there is only one artery perfusing both sides**. And even if only one

side is involved, only those tracts that decussate into the lesion (proximal in the tract, DCMLS from below the lesion, corticospinal tract from above) will be affected, and any decussations after the lesion (distal in the tract, DCMLS above the lesion, corticospinal tract below) will be spared.

Oh, and the outcome for most patients is excellent. An MRI shows the lesion, and unless it's vasculitis (typically embolic stroke), you treat it like a stroke, and the patient often does well—likely because the infarcted territory is so small, and either recanalization or angiogenesis restores perfusion. In case reports, most patients presented with symptoms far outside the tPA timeframe, so their condition resolved spontaneously.

So what people did was what is described in the next section: Assume the lesion is at the level that affects the hypoglossal nerve. That way, the corticospinal tracts have not yet decussated (which will present as complete contralateral hemiplegia, as the tracts that will decussate run through the lesion), and all of the DCMLS has decussated (which will present as complete contralateral hemisensory loss, as the tracts that have already decussated will run through the lesion), and the hypoglossal nerve can be compromised, even though **that isn't how it usually presents in real life**.

Anterior Spinal Artery for the Test

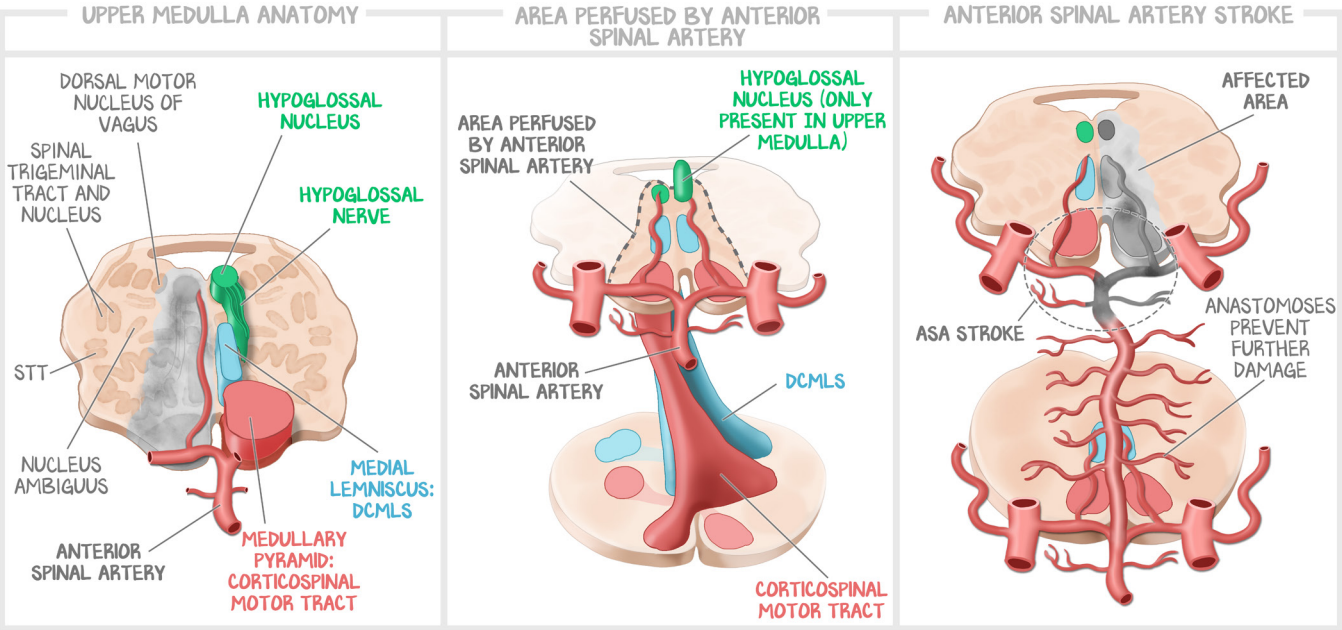

Figure 7.3: Anterior Spinal Stroke Faked for Ease
Inst-oh, magic-oh, change-oh! In the medulla—as seen specifically at the infinitesimal stretch of the open medulla with the fourth ventricle in it, superior to the decussation of the pyramids, superior to the DCMLS synapsing and crossing—the anterior spinal arteries (of which you know there is only one, but this is the sake of the test) are responsible for perfusing the ipsilateral corticospinal tract (which is also the pyramids), the ipsilateral medial lemniscus, and the ipsilateral hypoglossal nucleus and tract. A loss of the ipsilateral DCMLS will lead to perception deficits in the contralateral side of the body. A loss of the ipsilateral corticospinal tract will result in upper motor neuron symptoms in the entirety of the contralateral body. A loss of the ipsilateral hypoglossal nerve will cause the ipsilateral tongue muscles to be weak, and being unable to contract the ipsilateral muscles, tongue protrusion will result in a pull to the contralateral side.

Because ASA strokes of the medulla demonstrate the difference between tracts and nuclei so well, the "full" syndrome is what often ends up on licensing exams. The **loss of a nucleus** results in **ipsilateral findings**. The **loss of a tract** results in **contralateral findings**. According to the illustration below, loss of the ASA will compromise the **medial lemniscus** (DCMLS), the **corticospinal tract**, and the **hypoglossal nerve**. By this point in the medulla, no axon of the corticospinal tract has crossed, and so the whole tract runs through the lesion. Losing the whole tract before it crosses results in **contralateral hemiplegia** (an **upper** motor neuron symptom). By this point in the medulla, the DCMLS fibers have already crossed and are ascending into the lesion, resulting in **contralateral hemisensory loss**. Because the hypoglossal nucleus is lost, there is **ipsilateral paralysis**, which results in **tongue deviation** towards the **ipsilateral side** of the lesion.

For your test: **contralateral sensory loss**, **contralateral upper motor neuron**, and **ipsilateral hypoglossal dysfunction**.

Lateral Medullary—PICA

This one makes more sense than ASA stroke. PICA strokes usually result from a **thrombosis** or **embolism** that occludes the vertebral artery that gives rise to the PICA. Thrombosis, embolism, and dissection are the most common means of injuring the PICA itself.

The PICA branches off before the anterior spinal artery, travels laterally and superiorly along the medulla, and then crosses the inferior cerebellar peduncle. A stroke of the PICA affects the most superior medulla and inferior cerebellum. The AICA branches from the basilar artery, far from the origination of the PICA, but they both cross the inferior cerebral peduncle, bringing their vascular territories very close together. Some of the lateral medulla and lateral pons are perfused by branches from the basilar arteries, AICA, or PICA. With so much almost-redundant vasculature (but no anastomoses) and normal anatomic variation, patients often do not present with the full syndrome. As with ASA syndrome, you will be given the complete syndrome if you are expected to deduce the vessel in question. But you should be able to recognize the entire syndrome so that when you see parts of it, you know what else to look for.

At the top of the medulla is the **open medulla**, where the bottom of the fourth ventricle begins to emerge. The structures lost are the **vestibulocochlear nuclei**, **inferior cerebellar peduncle**, one of the **pain/temperature nuclei of CN V** (remember, its sensory column was the entire length of the brainstem), the **spinothalamic tract** (STT), **descending sympathetic fibers** (sympathetic fibers must run down through the brainstem to the first sympathetic ganglion in the trunk at C1 before ascending to the face, producing **ipsilateral Horner's**), and **nucleus ambiguous**.

Unilateral lesions of the **nucleus ambiguous** impair the function of CN IX and X, impairing the ability to input sensory information (CN IX), thus hindering motor signal output (CN X) during the swallowing and gag reflexes. There will also be **ipsilateral** paralysis of some of the muscles of the soft palate, larynx, and pharynx. There will be **displacement** of the uvula away from the side of the lesion (the good side pulls; the lesioned side cannot pull). The symptoms associated with loss of the nucleus ambiguous are **dysphagia**, **dysarthria**, and an **impaired gag reflex**. Although hoarseness can occur, speech is usually intelligible but difficult for the person to perform.

These patients typically complain of vestibular symptoms—vertigo, vomiting, nystagmus—and present with the cerebellar symptoms most associated with the trunk and upper limbs—veering or leaning to one side. The discoordination can be demonstrated with rapidly repeating movements (hand turn) producing dysdiadochokinesia. Alternatively, asking patients to drop their hands from a raised position and stop short at their lap, or raise their arms quickly and stop at eye level, can reveal defects due to an inferior cerebral peduncle lesion. The side with the defect will fail to stop.

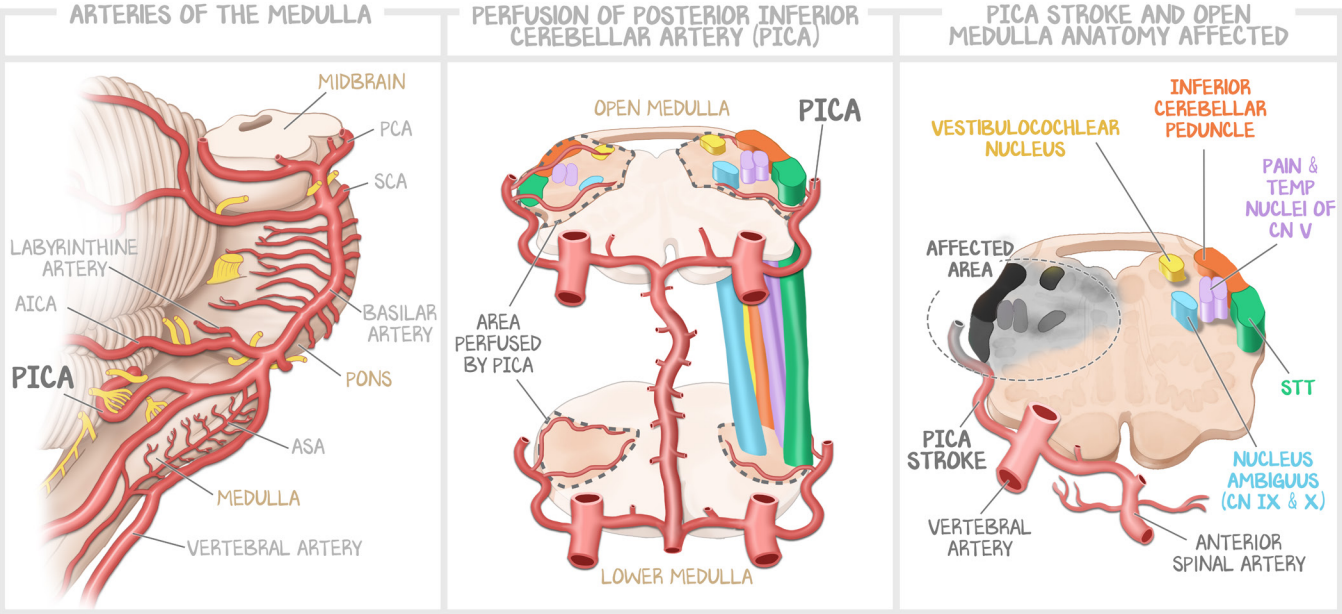

Figure 7.4: PICA Strokes
The PICA branches inferiorly but rises to perfuse the vestibulocochlear nucleus, inferior cerebellar peduncles, spinothalamic tracts (ipsilateral face, contralateral body), and descending sympathetics. PICA strokes are nearly identical to AICA strokes in presentation, except the PICA perfuses the nucleus ambiguous and not the facial nerve. Rather than a drooping face (AICA stroke), PICA strokes will present with speech and swallowing problems and uvula deviation (away from the side with the lesion).

STRUCTURE LOST	SYMPTOMS
Nucleus ambiguous	Dysphagia, dysarthria, impaired gag reflex Uvula deviation away from the lesion
Vestibulocochlear nuclei	Vomiting, vertigo, nystagmus
Inferior cerebellar peduncles	Ataxia on the ipsilateral side, falling Symptoms of peripheral vertigo
Pain/temperature nuclei	Ipsilateral loss of pain and temperature sensation from the face
Lateral spinothalamic tract	Contralateral loss of pain and temperature from all spinal levels from the body
Descending sympathetics	Horner's syndrome: miosis (constricted pupil unreactive to light), ptosis (droopy eyelid), and anhidrosis (no sweat on that side of the face).

Table 7.2: PICA Stroke Structures and Symptoms

Lateral Pontine—AICA

Even though the AICA and PICA originate far from one another, the structures at the top/rostral/superior medulla are the structures that make up the bottom/caudal/inferior pons. In AICA strokes, the nucleus ambiguous is not affected, so the patient is **spared dysphagia**. Instead, AICA strokes affect cranial nerve VII and **can** involve the labyrinthine artery.

Instead of the nucleus ambiguous, **cranial nerve VII** is completely lesioned. Loss of the facial nerve means the loss of motor function to half of the face, causing **ipsilateral facial droop** (including in the forehead) and **taste sensation loss** from the **ipsilateral anterior two-thirds** of the tongue. Although the facial nerve arises as two discrete nerves that quickly become one, both its motor nuclei and taste nucleus reside in the same vascular territory.

The labyrinthine artery sometimes branches from the basilar artery, but typically branches from the AICA. A stroke of the proximal AICA can, therefore, impair perfusion to the entire auditory apparatus, leading to **tinnitus** or even **deafness**. Although similar to the symptoms of damage to the vestibulocochlear nuclei (nausea, vertigo) seen with PICA strokes, the addition of total hearing loss on the affected side makes a lesion in the AICA more likely.

Furthermore, the cerebellar signs of middle cerebellar peduncle defects tend to lean more toward gait ataxia than truncal ataxia. You can't use that as a distinguishing feature because the AICA and PICA strokes are so variable, but in terms of separating them as syndromes, learn that PICA = trunk ataxia, AICA = leg ataxia.

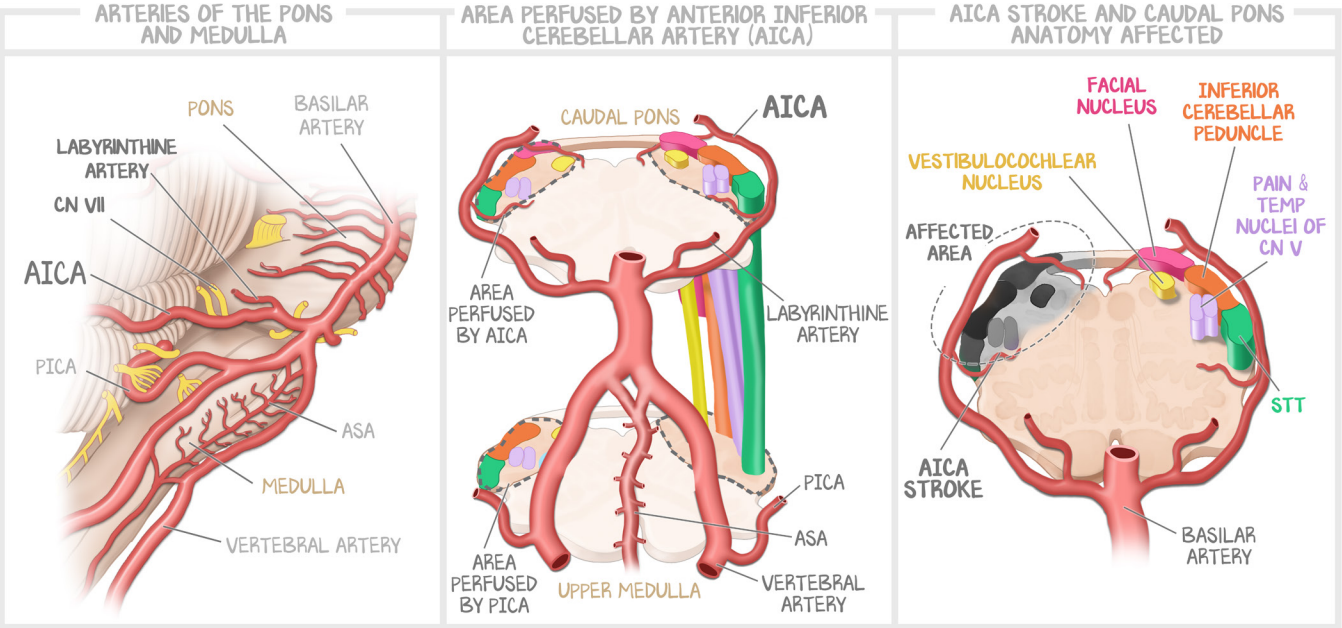

Figure 7.5: AICA Strokes
The AICA branches around the level of the medullary-pontine junction and wraps around the pons and cerebellum to perfuse the vestibulocochlear nucleus, inferior cerebellar peduncles, spinothalamic tracts (ipsilateral face, contralateral body), and descending sympathetics. The AICA is nearly identical to the PICA, except the AICA perfuses the facial nerve (cranial nerve VII), which provides motor innervation to the face. The loss of the AICA can cause all of the same symptoms as a loss of AICA, except that loss of the AICA will not cause any speech or swallowing deficiencies but will cause facial droop, instead.

STRUCTURE LOST	SYMPTOMS
CN VII	Facial droop and loss of taste in anterior 2/3 of tongue Loss pain and temperature sensation from face
Vestibulocochlear nuclei	Vomiting, vertigo, nystagmus
Inferior and middle cerebellar peduncles	Ataxic gait on the ipsilateral side, falling
Pain/temperature nuclei	Ipsilateral loss of pain and temperature sensation from the face
Lateral spinothalamic tract	Contralateral loss of pain and temperature from all spinal levels, from body
Descending sympathetics	Horner's syndrome: miosis (constricted pupil unreactive to light), ptosis (droopy eyelid), and anhidrosis (dry eyes).

Table 7.3: AICA Stroke Structures and Symptoms

Midbasilar Stroke—Locked-In Syndrome

One of the most devastating basilar artery occlusions is a midbasilar occlusion with **bilateral pontine ischemia**, an obstruction of the basilar artery itself, especially at a point where it gives rise to the median or paramedian branches into the pons. The collateral circulation for the superior pons comes from the anterior circulation. The collateral circulation for the inferior pons comes from the vertebral arteries. But at the segment with the lesion (usually an **embolism**), there is no collateral circulation. And if the embolism occludes a paramedian or median branch, the medial pons immediately below the lesion infarcts. It may be just a little infarct, a sliver of the pons that is lost. But because the basilar artery is one artery, **both sides are affected**. And the DCMLS, corticospinal tract, and corticobulbar tracts are all present in the pons. Losing **both sides of the DCMLS** means there can be no perception of any sensation below the level of the lesion—complete loss of touch sensation from the neck down. Losing **both corticospinal tracts** means the patient will have **bilateral upper motor neuron** symptoms of the entire body from the neck down. Losing **both corticobulbar tracts** means there are **bilateral upper motor neuron** symptoms to the cranial nerves below the lesion—all of them except those that are "midbrain or higher." CN I, II, III, and IV are spared. So, even though the cranial nerve nuclei are still intact, and the facial nerve can induce movement, and the accessory nerve can still shrug and turn the head, there is no cortical signal to their nuclei to induce them.

The patient cannot move their extremities, body, or face, or use their mouth—**total paralysis**. They have no ability to feel pressure, vibration, or proprioception. Depending on the level of the lesion, sensation may be retained in the face (trigeminal). Pain and temperature sensation are preserved. Cranial nerves II, III, and IV are usually intact. Cranial nerve III raises the eyelid. It is said that these patients communicate through blinking. No. The only way patients with this syndrome can communicate is by actively contracting their eyelid to open and letting it close. That is **harder than blinking**.

The patient can see, hear, smell, move their eyes (sort of; their lateral recti are not innervated, so there is a gaze defect), and open their eyelids. **Consciousness is fully intact.** This condition was commonly misdiagnosed as a **persistent vegetative state**—cerebral death (no consciousness, no purposeful interactions) but ongoing life (all corporeal bodily functions continue, including sleep-wake cycles and, in most cases, the return of infantile reflexes). **Locked-in syndrome** is the opposite. The brainstem is broken, but the cerebrum is fully intact.

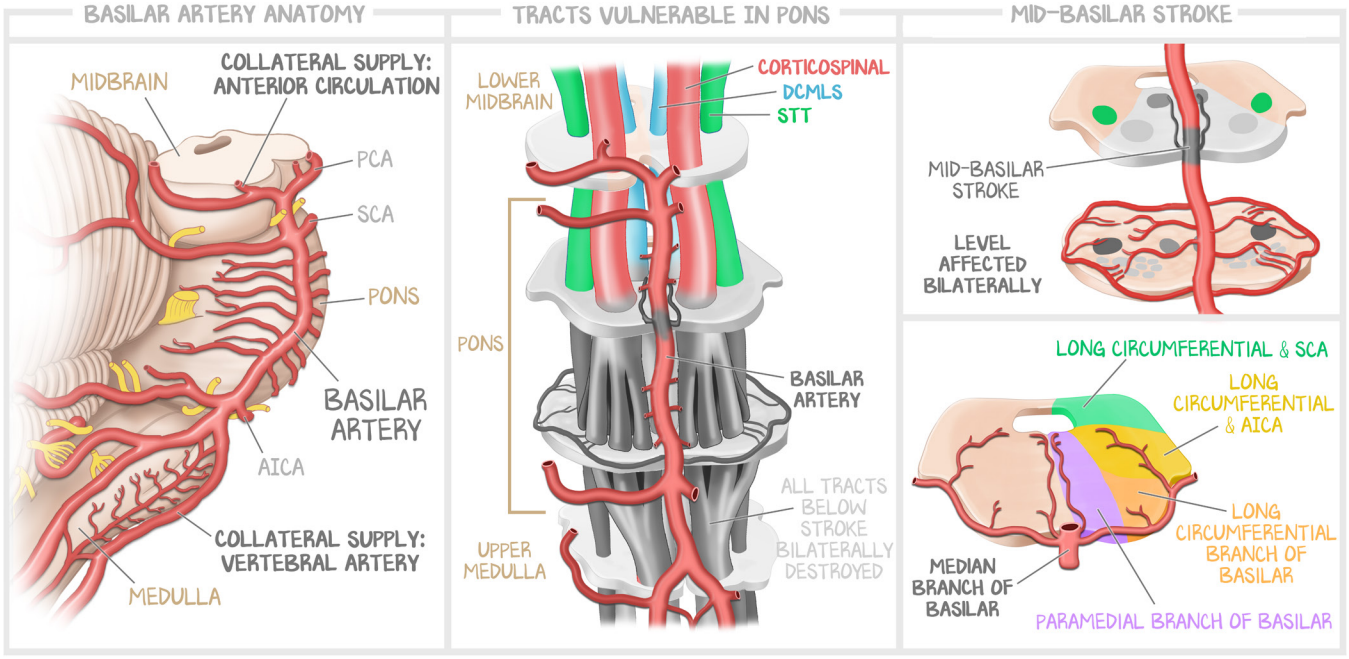

Figure 7.6: Midpontine Syndrome
Because the one pontine artery perfuses the entirety of the pons, and because tracts must be intact the entire length of their axons to transmit a signal, a stroke of the basilar artery can compromise the entirety of the sensory and motor connection from the head to the body, causing locked-in syndrome. What we have shown is a "total pons stroke," compromising the entire pons (all is grey). Because the posterior cerebral arteries provide an anastomosis from above, and the vertebral arteries provide an anastomosis from below, the basilar artery stroke must occur at exactly the level that the paramedian branches originate. It's devastating if it happens, but it's highly unlikely that it will.

Fortunately, this condition does not occur frequently. Any loss to neural tissue is irreparable. Whatever tissue is lost will never recover.

Cerebellar—Superior Cerebellar Artery (SCA)

The superior cerebellar artery perfuses the cerebellum from the superior cerebellar peduncle. Classic descriptions of this lesion include the symptoms of the rostral/top/superior pons, those that sound very similar to the PICA and AICA strokes (particularly the loss of tracts, such as the LSTT and descending parasympathetics). However, most patients present with **cerebellar signs only**—minor vertigo (the vestibulocochlear nucleus and nerve are not affected), **dysarthria** (most common, mouth discoordination), limb discoordination, **dysmetria** (overshoot or undershoot with their finger when trying to touch something), and gait ataxia with veering.

SCA strokes are almost always embolic.

Weber Syndrome

The Weber listed in all those syndromes that you don't like studying was the son of a German physician who defined this syndrome, Hermann David Weber. His son, Frederick Parkes Weber, while defining the many syndromes that bear his name in London, he retained his family's German connections, as emphasized by retaining the continental "V" sound in his surname. All the fighting over whether it's "WEE-bur" or "Webber" is for naught. Some common arguments often arise. It isn't regional dialect or "*how I was taught it*" (ask any resident or even MS4 how it's said). "Veh-buh" (veh as in vein, buh as in butter) is the closest approximation in English.

And alas, it is an eponym, and so it's better termed **superior median midbrain syndrome**. It is caused by the occlusion of the **paramedian branch** of the basilar artery just prior to bifurcating into the posterior cerebral arteries. This is no different from the branches of the basilar artery that perfuse the pons, but these latter branches perfuse the medial midbrain.

At the level of the superior colliculus, the paramedian artery perfuses the **medial lemniscus**, **oculomotor nerve**, **oculomotor nucleus**, **Edinger-Westphal nucleus**, and the medial portion of the **crus cerebri**. Thus, symptoms include **contralateral hemisensory loss** (upper extremity, lower extremity, and face) due to the loss of the medial lemniscus (DCMLS), **ipsilateral oculomotor palsy** due to loss of the oculomotor nucleus, **ipsilateral miosis** due to the loss of the E-W nucleus, and some **contralateral** upper motor neuron lesions of the corticobulbar tract, as they run the most medially in the crus cerebri. Some corticospinal axons can be affected, as well.

A variant of Weber syndrome is Benedikt syndrome. This is better named **inferior median midbrain syndrome**. At the level of the inferior colliculus, the cerebellar pathways to and from the cortex decussate. The same lesion in the paramedian branch could affect this region. Here, there is much more **hemiataxia** than hemiplegia.

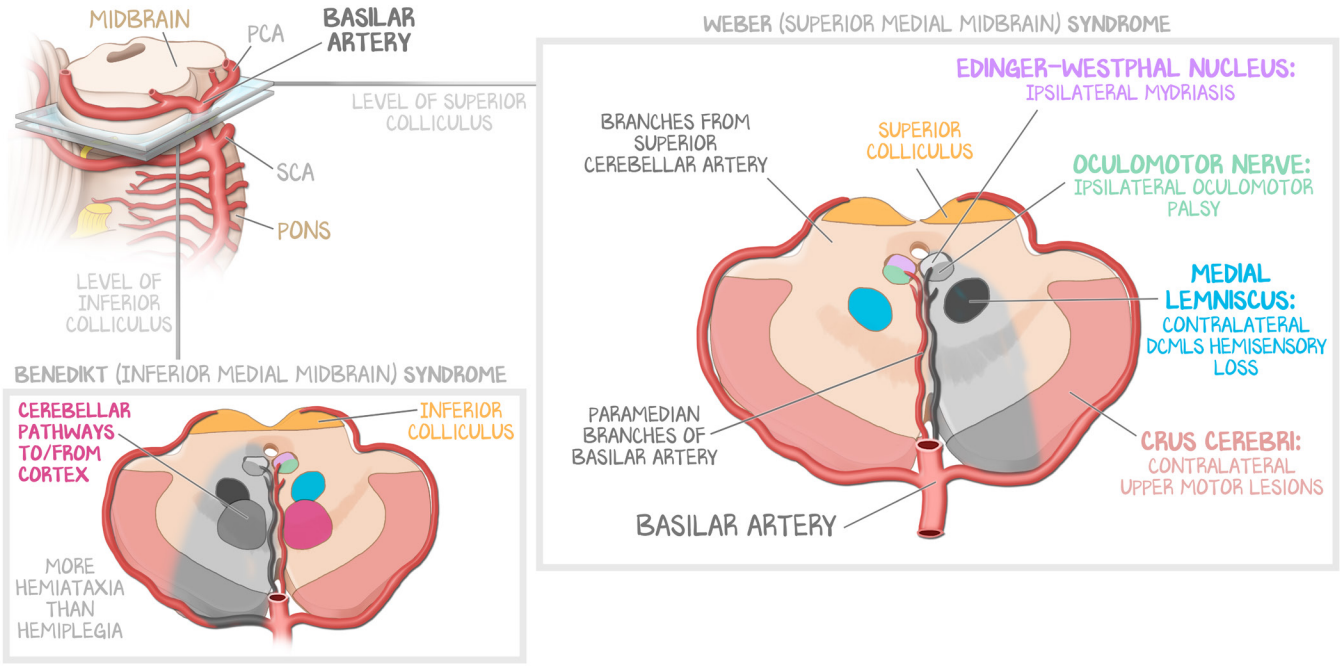

Figure 7.7: Medial Midbrain Lesions
Weber syndrome is caused by a superior medial midbrain stroke, in which there are compromise of the medial lemniscus (loss of contralateral general sensation), crus cerebri—the corticospinal tract (loss of contralateral motor) and the nuclei of the oculomotor nerve—and both Edinger-Westphal and Oculomotor nuclei (ipsilateral eye muscle palsy—special senses, later this module—and a dilated pupil).